(FILE 'HOME' ENTERED AT 09:10:35 ON 14 MAY 2003)

FILE 'REGISTRY' ENTERED AT 09:11:00 ON 14 MAY 2003

L1 320 S NEOMYCIN

FILE 'MEDLINE, CAPLUS, BIOSIS' ENTERED AT 09:14:44 ON 14 MAY 2003

L2 21562 S L1

L3 28309 S NEOMYCIN OR NEAMINE OR NEOBIOSAMINE

L4 32409 S L2 OR L3

L5 3239 S L4 AND (ANTIVIRAL OR VIRUS)

L6 103 S L5 AND REVERSE (W) TRANSCRIPTASE

L7 90 S L6 AND PY<= 2001

L8 50 DUPLICATE REMOVE L7 (40 DUPLICATES REMOVED)

=> s DNA (w) hybrid

L9 2825 DNA (W) HYBRID

=> s RNA (w) hybrid

L10 2047 RNA (W) HYBRID

=> s 19 or 110

L11 4529 L9 OR L10

=> s 18 and 111

L12 0 L8 AND L11

=> 111 and 14

L11 IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system. For a list of commands available to you in the current file, enter "HELP COMMANDS" at an arrow prompt (=>).

=> s 111 and 14

L13 8 L11 AND L4

=> duplicate remove 113

DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS'

KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n

PROCESSING COMPLETED FOR L13

L14 7 DUPLICATE REMOVE L13 (1 DUPLICATE REMOVED)

=> d ibib abs 1-7

L14 ANSWER 12 OF 30 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER:

2001:177423 CAPLUS

DOCUMENT NUMBER:

135:40492

TITLE:

Aminoglycoside antibiotics, neamine and its

derivatives as potent inhibitors for the RNA-protein

interactions derived from **HIV-1** activators

AUTHOR (S):

Hamasaki, K.; Ueno, A.

CORPORATE SOURCE:

Department of Bioengineering, Graduate School of Bioscience and Biotechnology, Tokyo Institute of

Technology, Yokohama, 226-8501, Japan

SOURCE:

Bioorganic & Medicinal Chemistry Letters (2001),

11(4), 591-594

CODEN: BMCLE8; ISSN: 0960-894X

PUBLISHER:

Elsevier Science Ltd.

DOCUMENT TYPE:

Journal

LANGUAGE:

English

18

Neamine derivs. which have an arginine (RN), a pyrene (PCN) and both pyrene and arginine (PRN) have been prepd. and their binding toward the RNA fragments derived from HIV-1 activator region, TAR and RRE RNA were examd. Among them, PRN bound either TAR RNA or RRE RNA with equiv. binding affinities as Tat and Rev peptide, resp. Neamine derivs. which have an arginine (RN), a pyrene (PCN) and both pyrene and arginine (PRN) have been prepd. and their binding toward the RNA fragments derived from HIV-1 activator region, TAR and RRE RNA was examd. Among them, PRN bound either TAR RNA or RRE RNA with equiv. binding affinities as Tat and Rev peptide, resp.

REFERENCE COUNT:

THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 21 OF 30 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER:

1998:275253 CAPLUS

DOCUMENT NUMBER:

128:316931

TITLE:

Binding of Neomycin to the TAR Element of HIV -1 RNA Induces Dissociation of Tat Protein by an

Allosteric Mechanism

AUTHOR(S):

Wang, Shaohui; Huber, Paul W.; Cui, Mei; Czarnik,

Anthony W.; Mei, Houng-Yau

CORPORATE SOURCE:

Department of Chemistry and Biochemistry, University

of Notre Dame, Notre Dame, IN, 46556, USA Biochemistry (1998), 37(16), 5549-5557

CODEN: BICHAW; ISSN: 0006-2960

PUBLISHER:

SOURCE:

American Chemical Society

DOCUMENT TYPE:

Journal

LANGUAGE: English

Neomycin inhibits the binding of Tat-derived peptides to the trans-activating region (TAR) of HIV-1 RNA. Kinetic studies reveal that neomycin acts as a noncompetitive inhibitor that can bind to the Tat-TAR complex and increase the rate const. (koff) for dissocn. of the peptide from the RNA. Neomycin effects a conformational change in the structure of TAR that can be detected by CD spectroscopy. The increase in ellipticity measured at 265 nm upon binding of the aminoglycoside is opposite to the decrease seen when Tat peptides bind to the RNA. Thus, the structural transition induced by neomycin is apparently incompatible with the binding of Tat and underlies the inhibitory action of the antibiotic. The binding site for neomycin on TAR was identified in RNase protection expts. and is located in the stem immediately below the three-nucleotide bulge that serves as the primary identity element for Tat. Apparent protection of residues in the bulge by neomycin may represent addnl. contacts to the aminoglycoside, but more likely result from changes in the structure of this region when the ligand binds to the RNA. Binding assays using variants of TAR in which inosine residues were substituted for guanosine residues support the results from the RNase protection expts. Inosine substitutions in the lower stem, but not the upper stem, decrease the binding const. for neomycin by approx. 100-fold. Neither of these variants affected the binding affinity of Tat peptide. In addn., these latter expts. suggest that the aminoglycoside may be located in the minor groove of the stem. This mode of assocn. may be a crit. aspect of neomycin's ability to bind to the Tat-TAR complex and could serve as a guide for the design of other drugs that bind to specific RNA targets as noncompetitive inhibitors.

L14 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2001:745118 CAPLUS

DOCUMENT NUMBER: 136:33494

TITLE: Neomycin-Induced Hybrid Triplex Formation

AUTHOR(S): Arya, Dev P.; Coffee, R. Lane, Jr.; Charles, I. CORPORATE SOURCE: Laboratory of Medicinal Chemistry Department of

Chemistry, Clemson University, Clemson, SC, 29634, USA

Journal of the American Chemical Society (2001),

123 (44), 11093-11094

CODEN: JACSAT; ISSN: 0002-7863

PUBLISHER: American Chemical Society

DOCUMENT TYPE: Journal LANGUAGE: English

SOURCE:

AB Recent work has shown the remarkable ability of aminoglycosides (in particular, neomycin) to stabilize RNA and DNA triple helixes.

The triplex stabilization effect of neomycin was shown to be the highest among all groove binders previously studied (which tend to prefer the duplex structures). In our quest to expand the triplex stabilization potential of neomycin, we report the remarkable ability of neomycin to induce hybrid DNA-RNA-DNA as well as DNA-RNA-RNA triplexes. Aminoglycosides are also shown to stabilize the hybrid DNA-RNA duplex.

REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 13 OF 26 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1999:502766 CAPLUS

DOCUMENT NUMBER: 131:153725

TITLE: Small molecule inhibition of RNA/ligand binding

INVENTOR(S): Green, Michael R.; Zapp, Maria L.

PATENT ASSIGNEE(S): University of Massachusetts Medical Center, USA

SOURCE: U.S., 14 pp.

CODEN: USXXAM Patent

DOCUMENT TYPE:

LANGUAGE: English

FAMILY ACC. NUM. COUNT:

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5935776	Α	19990810	US 1995-399378	19950302
US 5534408	Α	19960709	US 1993-126236	19930924
PRIORITY APPLN. INFO.	:		US 1992-965341	19921023
			US 1993-126236	19930924

A method is disclosed for the inhibition of binding of a ligand to an RNA, AB the inhibition being mediated by a small org. mol. that binds to the RNA, thereby inhibiting ligand binding. The invention is particularly directed to the interaction of the Rev protein of HIV with the Rev-responsive element (RRE) present in HIV-derived mRNA mols. A preferred class of small org. mols. are compds. exemplified by 2,5-Bis[4-(2-N,Ndimethylaminopropylamidino)phenyl]furan.

REFERENCE COUNT: THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS 16 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT L10 ANSWER 12 OF 26 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1999:736476 CAPLUS

DOCUMENT NUMBER: 131:346535

TITLE: Use of neomycin for treating angiogenesis-related

diseases

INVENTOR(S): Hu, Guo-Fu; Vallee, Bert L.

PATENT ASSIGNEE(S): The Endowment for Research In Human Biology, Inc., USA

SOURCE: PCT Int. Appl., 74 pp.

CODEN: PIXXD2

DOCUMENT TYPE:

LANGUAGE:

Patent English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

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PATENT NO.
                  KIND DATE
                                       APPLICATION NO. DATE
                                        ______
    WO 9958126
                    A1 19991118
                                       WO 1999-US10269 19990511
        W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ,
            DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS,
            JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,
            MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,
            TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ,
            MD, RU, TJ, TM
        RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
            ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,
            CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
                                       CA 1999-2331620 19990511
    CA 2331620
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                    AA
    AU 9939804
                     A1
                          19991129
                                        AU 1999-39804
                                                         19990511
    EP 1083896
                     A1
                                       EP 1999-922915
                          20010321
                                                         19990511
          AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, FI
                          20021119
                                                         20001109
    US 6482802
                     B1
                                        US 2000-700436
PRIORITY APPLN. INFO.:
                                      US 1998-84921P P 19980511
                                      WO 1999-US10269 W 19990511
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AB The present invention is directed to using neomycin or an analog thereof as a therapeutic agent to treat angiogenesis-related diseases, which are characterized by excessive, undesired or inappropriate angiogenesis or proliferation of endothelial cells. The present invention is also directed to pharmaceutical compns. comprising: (a) neomycin or an analog and, optionally, (b) another anti-angiogenic agent or an anti-neoplastic agent. The present invention is further directed to a method for screening neomycin analogs having anti-angiogenic activity. A preferred embodiment of the invention relates to using neomycin to treat subjects having such diseases. A dose of 20 ng neomycin/embryo or higher completely inhibited angiogenin-induced angiogenesis in the chorioallantoic membrane (CAM) assay. Neomycin inhibits angiogenin-induced angiogenesis mainly through inhibition of nuclear translocation of angiogenin.

REFERENCE COUNT: 1 TI

THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 9 OF 30 MEDLINE

ACCESSION NUMBER: 2001609883 MEDLINE

DOCUMENT NUMBER: 21540632 PubMed ID: 11684311

TITLE: Anti-HIV activity of a novel aminoglycoside-

arginine conjugate.

AUTHOR: Cabrera Cecilia; Gutierrez Arantxa; Barretina Jordi; Blanco

Julia; Litovchick Alexander; Lapidot Aviva; Clotet

Bonaventura; Este Jose A

CORPORATE SOURCE: Retrovirology Laboratory, Fundacio irsiCaixa, Hospital

Universitari Germans Trias i Pujol, Universitat Autonoma de

Barcelona, 08916, Badalona, Spain.

SOURCE: ANTIVIRAL RESEARCH, (2002 Jan) 53 (1) 1-8.

Journal code: 8109699. ISSN: 0166-3542.

PUB. COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200203

ENTRY DATE: Entered STN: 20011102

Last Updated on STN: 20020307 Entered Medline: 20020305

We have previously described conjugates of L-arginine with aminoglycosides (AAC) that have shown anti-human immunodeficiency virus type 1 (HIV-1) activity in in vitro cell culture systems. Here, we extend our report to a novel neomycin B-arginine conjugate (NeoR) that has shown up to 30-fold increased potency over previous AAC compounds. NeoR inhibited the replication of both R5 and X4 strains of HIV-1 in cells expressing the appropriate coreceptor or peripheral blood mononuclear cells. In lymphoid tissue ex vivo, NeoR blocked the replication of the dualtropic strain 89.6 suggesting anti-HIV activity of AAC on the site of in vivo virus replication. NeoR blocked the binding of HIV particles to lymphoid cells and was also able to antagonize the activity of the CXCR4 receptor so it may prevent the emergence of X4 HIV-1 strains. Nevertheless, in a cellular assay, we were unable to detect anti-Tat dependent transactivation activity as previously suggested for this family of compounds.

L10 ANSWER 23 OF 26 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: DOCUMENT NUMBER:

1996:408688 BIOSIS

PREV199699131044

TITLE:

Ligands of the antiestrogen-binding site are able to

inhibit virion production of human immunodeficiency virus

1-infected lymphocytes.

AUTHOR(S):

Mesange, F.; Delarue, F.; Puel, J.; Bayard, F.; Faye, J.-C.

CORPORATE SOURCE:

(1) Lab. Endocrinol. Communication Cellulaire, INSERM U397,

Inst. Louis Bugnard, CHU Ranqueil, Ave. J. Pulhes, 31045

Toulouse Cedex France

SOURCE:

Molecular Pharmacology, (1996) Vol. 50, No. 1, pp. 75-79.

ISSN: 0026-895X.

DOCUMENT TYPE:

Article English

LANGUAGE:

Since the discovery of human immunodeficiency retrovirus, the drug arsenal against retrovirus has rapidly increased. Concomitantly, new challenges in the therapy of acquired immune deficiency syndrome have arisen, including drug toxicities, drug resistance, and the development of various cancers as effective therapies prolong survival. Tamoxifen, a nonsteroidal antiestrogen with a low incidence of side effects, is widely used in cancer therapy; it is known to exert pleiotropic activities by binding essentially to the estrogen receptor and other unidentified proteins. In the present work, quantification of the p24 core protein of human immunodeficiency virus 1 produced by infected lymphocytes shows an inhibitory effect of tamoxifen on virion production. Moreover, we assume that this effect is not mediated by the estrogen receptor because antiestrogen ligands interacting with the antiestrogen-binding site exhibit efficacy related to their affinity for this site, although specific antiestrogens of the estrogen receptor are ineffective.